AMENDMENTS TO THE SPECIFICATION:

Please replace the paragraphs at page 4, lines 1-18 with the following replacement paragraphs:

(for review, see Selkoe et al., Nature 399(6738 Suppl):A23-31 (1999); Tekirian, J. Alzheimers. Dis. 3(2):241-248. (2001)). The APP sequence near the β-secretase cleavage site is:

EVKM*DAE. (SEQ ID NO: 34)

These residues are labeled P4-P3-P2-P1*P1′-P2′-P3′ in standard protease nomenclature with the cleavage site between P1 and P1′ marked by *. Mutations in this region, such as the KM to NL mutation (the so-called Swedish mutation), can transform APP into a more preferred substrate for BACE. Hence, amino acid sequence changes in APP that result in increased APP cleavage by BACE increase the likelihood of the development of Alzheimer's (Citron et al., Nature 360(6405): 672-674 (1992)).[[.]] Experimental evidence suggests that APP processing is sequential and that cleavage of APP by beta-secretase is a prerequisite for gamma–secretase-mediated APP processing. Cleavage within the transmembrane region of APP by gamma-secretase results in the 40/42- residue Aβ peptide, whose elevated production and accumulation in the brain are the central events in the pathogenesis of Alzheimer's disease (Selkoe,. Nature 399:23-31 (1999)). In addition, it is now clear that BACE can again cut Aβ peptide 40-42 after gamma-secretase to generate a neurotoxic Aβ34 peptide, at the expense of Abeta40-42 (Fluhrer et al., J. Biol. Chem. 278(8): 5531-5538 (2003)).